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Editor:
FREDERICK HOBDAY, C.M.G., F.R.C.V.S., F.R.S.E.,
HONORARY VETERINARY SURGEON TO HIS MAJESTY THE KING,
OFFICIER DU MÉRITE AGRICOLE (FRANCE),
CAVALIERE DEI SS. MAURIZIO E LAZZARO (ITALY),
HONORARY MEMBER OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION,
AND FORMERLY PROFESSOR IN THE ROYAL VETERINARY COLLEGE, LONDON.

Sub-Editor:
GLADSTONE MAYALL, M.R.C.V.S.

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Editorial

THE VALUE OF AN EFFICIENT GOVERNMENT VETERINARY DEPARTMENT.

No better illustration could be given as showing the value of an efficient and scientific veterinary department where the diseases of animals are concerned than by drawing attention to the fact that, although Great Britain has been for some time free from rabies, during the past twelve months two cases of this terrible disease have occurred in imported dogs whilst undergoing the prescribed term of quarantine. In one of the instances the animal, which was brought from Egypt, showed signs of rabies on the third day after its arrival, and upon tracing its history back it was found that it had been bitten by a rabid animal some two months previously. In the second case the symptoms did not appear until the dog had been in quarantine in England for three months; so that when this is added to the length of time taken by the journey, it must have received the infection at least four months before becoming rabid.

Had these dogs not been under proper veterinary supervision, Great Britain would have become re-infected, and there is no telling what might have happened. They are distinct illustrations of the

value of the employment, when drafting up preventive regulations against the diseases of animals, of those who possess expert knowledge of the subject ; and where these diseases are concerned the only competent adviser whose claims can legitimately be put forward in this category is the duly qualified and scientific veterinarian.

General Articles

EAST COAST FEVER—THE THEORY OF LATENCY.*

By Ll. E. W. BEVAN, M.R.C.V.S.,
Director of Veterinary Research, Southern Rhodesia.

Since 1901, the disease known as East Coast fever of cattle has seriously handicapped the progress of the pastoral industry in Southern Rhodesia, not only by the loss of cattle directly or indirectly attributable to it, but by the restriction of movement and transport and general interference with business. Although, as the result of the investigations of Koch, Theiler, Watkins-Pitchford, and others, the cause of the disease and its method of transmission were rapidly determined, and scientific means of control by systematic dipping devised, the disease persists and outbreaks continue to occur. It frequently happens that it makes its appearance in areas where the disease previously existed, and in herds containing the survivors of previous outbreaks, in circumstances which would appear to exclude any association with existing outbreaks.

Notwithstanding the difficulty of excluding the possibility of illicit movement of cattle, or the accidental transportation of ticks in hay, forage, kaffir blankets, or railway-trucks, there are many who have had experience of the disease, including several members of the veterinary profession, who attribute such recrudescences to infection derived from an animal which, having recovered during a previous outbreak, has retained the parasite in a dormant or latent form. The exponents of this theory, however, do not demonstrate in what form the parasite persists in the recovered animal, nor do they explain how it is that an animal " carrying " such a parasite is for long periods non-infective but suddenly becomes a source of danger. There are some who consider that the infection is carried on through the calves, but do not explain how this is brought about. Much has been said and written on the theory, but, as far as we know, no scientific evidence has yet been advanced to prove it : it

* Paper presented at a Meeting of the Royal Society of Tropical Medicine and Hygiene, and published in the *Transactions* of that Society, vol. xviii.

is based entirely upon what is known as practical experience. Others, on the other hand, basing their opinion on the observations and deductions of Gonder (1), (2), (3), consider that in animals which recover the parasite perishes and a complete sterile immunity is established: and consequently that there can be no relapse and no possibility of infecting new ticks.

In order better to understand Gonder's description and the life-cycle of the causal organism of the disease—*Theileria parva*—and the arguments upon which these contensions are based, it may be well first to discuss briefly some of the characteristics of the protozoa in relation to disease. The protozoa are unicellular animal organisms, forming the lowest division of the animal kingdom, many of which are saprophytic, but others are parasitic and entirely dependent upon some particular organism or group of organisms for their existence. Some can live in harmony with their host, but others produce harmful effects and give rise to disease in it. It is, obviously, to the disadvantage of the parasite to bring about the death of its host, unless provision is made for its transference to another. Such transference may take place by the mechanical transmission of resistant forms of parasites, liberated by the breaking down of the tissues after death or in discharges from ulcers or infected mucous membranes during life, or the parasite may be taken up from the infected animal by an alternate host, frequently an invertebrate, in which it establishes itself until it can be transferred once more to a susceptible animal in which to complete the cycle of development. As the period, in either one or the other host, may be brief or lengthy, the life-cycle of the organism is adapted to the special conditions which may present themselves. The reproduction of such organisms is by fission, and in some instances multiplication can take place almost indefinitely without syngamy: the sexual act appears to be only necessary to restore vitality. East Coast fever is caused by such a protozoal parasite—*T. parva*—which is transmitted from sick to healthy animals by ticks belonging to the species Rhipicephalidæ.

In the Annual Reports of the Union of South Africa, Department of Agriculture, 1909–11, Gonder describes its development as follows: "... the minute sporozoites injected by the tick collect in the spleen and lymphatic glands, where they penetrate into the lymphocytes, in which they grow rapidly. The originally single nucleus divides repeatedly, and large multinucleate plasmodial masses are formed which finally divide up into as many minute merozoites, *agamonts*, as there are nuclei; the process recalls strongly the schizogony of *Hæmoproteus columbæ*, and leads to the breaking up of the lymphocyte. The first schizogonous generation may be

repeated several times, but at last a generation of gamonts is produced, which are distinguished from the agamonts by characteristic differences in the nuclear structure. The gamonts multiply by a process of schizogony, the final, or *gamogonous*, generation ending in the production of gametocytes, minute parasites which do not multiply further, but penetrate into the red blood corpuscles, where they grow into adult gametocytes of two kinds—male gametocytes, which are long, slender, bacillary forms; and female gametocytes, which are plump, rounded, or pear-shaped forms. The gametocytes can only develop further in the tick." "The forms found in the red corpuscles in the peripheral blood are either gamonts or gametocytes, incapable of developing beyond the latter stage except in the tick; this explains a peculiarity of this parasite, namely, that inoculation of infected blood into a healthy animal does not produce an infection."

The further development of the *T. parva* in the tick has also been described by Gonder: "Within an hour after passing into the stomach of the tick the parasites become free from the corpuscles. The immature gametocytes die off, but the adult forms proceed to gamete-formation. The free parasites are at first rounded off, but soon send out processes and become amœboid. The male gametocytes send out a single process, and creep about actively like a limax-amœba; their nucleus goes through an unequal division, after which the gametocyte becomes a gamete. The female gametes, which are inactive, go through a similar reduction-process. Pairing of two gametes and fusion of the cytoplasm bodies takes place, but before the nuclei fuse each nucleus goes through a second reduction division. After copulation of the nuclei the zygote becomes an active oökinete, first retort-shaped and then gregarinform, which penetrates into the salivary glands, and there goes through a multiplicative process, very similar to that of *Halteridium* in the lung of a pigeon, producing a swarm of sporozoites which are inoculated into the vertebrate host by the tick."

In the report for 1909–10, page 75, Gonder states: "It is a fact that no parasites *are to be discovered* in so-called salted cattle, i.e. immune against East Coast fever, and that the latter have, *up till now*, always been found not to be subject to relapse, *which proves that the parasites must perish after a certain period, and that they cannot undergo parthenogenesis.* It is also impossible to transmit East Coast fever by means of blood, a circumstance which speaks against a further development of the parasites in the blood. Cattle recovering from the disease show agamogonous and gamogonous forms if the disease is at its height. The agamogonous forms disappear very soon. If the number of parasites decrease in the blood, the number

of gamogonous forms will lessen in the organs, i.e. if the gamogonous forms disappear in the organs, lymphatic glands, spleen, etc., the development of the parasites in the body of the animal is arrested."

Again, on page 77 of the same report, he says: " . . . the agamete produced by schizogony out of an agamont can again become an agamont, or, in other words, the agamogonous development repeats itself. However, the parasites, like all protozoa (after a certain time, or, rather, after the agamogonous development) have to undergo a regulating process. This latter process will enable it to start a new life, which is due to fertilisation. We are accustomed to a change of hosts in the protozoic blood parasite, which in the course of its phylogenetic development has adapted itself suitably to its respective parasite. The parasite completes in one host its agamogonous and progamogonous stages until it has developed into gametocytes, and in the second host finishes up with its sexual, gamogonous, and postgamogonous development. Besides this development we are aware of parthenogenesis, a process of fertilisation which enables the female forms to obtain agamogonous forms by special metamorphosis of the nucleus. *T. parva* is an exception in this case. *Parthenogenesis does not occur, consequently there is no relapse and no possibility of infecting new ticks,* after the animal has recovered."

In an article submitted to the *Journal of Comparative Pathology and Therapeutics*, December 1910, p. 332, he says: " With the formation of the gametocytes the cycle of *T. parva* in an animal arrives at a definite conclusion, and the animal either recovers or dies. In cattle which recover from the disease a general decrease of the parasites is noted in the blood after the crisis. The agamogonous forms disappear, and with this the fever gradually subsides. The gamogonous stages do not develop beyond the formation of gametocytes, and these are the endoglobular parasites. *Since we do not find parthenogenesis, the animal is completely protected against relapses, and recovery leaves a complete sterile immunity. No tick can infect itself on this animal, and no infected tick can infect such a beast.*"

The scientific view that the so-called " salted " animal cannot be a source of danger is largely based upon the foregoing observations and deductions which, being somewhat confusing, may be summarised as follows :—

1. Because no parasite can be found in a recovered animal, and because the recovered animal has not been found to be subject to relapse, therefore the parasites must perish.

2. Because parthenogenesis does not occur the animal is completely protected against relapse.

3. And, conversely, because the recovered animal is not subject

to relapse, the parasite must perish after a certain period, and cannot undergo parthenogenesis.

4. Finally, because there is no relapse, there is no possibility of infecting new ticks after the animal has recovered.

A careful analysis of the above indicates that in more than one respect Gonder has been guilty of "begging the question." Also that his deductions may not be entirely accurate is suggested by Minchin's criticism of his description of the development of *T. parva* in which he writes: " . . . it is a pity that the effect of such excellent work should be marred by so much theoretical bias. Aber wie die Alten sungen. . . ." (4)

In the first place, Gonder takes for granted that the recovered animal does not relapse, and bases some of his deductions upon this assumption. The argument appears to be that because the recovered animal is not subject to a relapse the parasite must perish and therefore cannot undergo parthenogenesis, and because parthenogenesis does not occur the animal is completely protected against relapse.

The failure to detect the parasite in a recovered animal does not prove that it has perished; it may be present in some unrecognised form or in some organ which has not been sufficiently examined. It is well known that in acute cases of piroplasmosis of cattle and dogs the parasite is numerous in the peripheral blood and internal organs, but on recovery cannot be found. The most exhaustive search of the blood and tissues of a recovered animal would at most times fail to reveal a single parasite. But that they are present is shown by the fact that if a drop of blood from a recovered animal is inoculated into a suceptible one it will give rise to an acute infection and possibly death. Similarly, it may be impossible to detect trypanosomes in infected game, but their blood is infective.

The argument that the "parasite must perish" is also based upon the assumption that the recovered animal does not relapse, and that the disease cannot be transmitted by blood inoculation. This, however, cannot be accepted. The disease known as Egyptian fever (5), which is also a theileriasis, cannot be transmitted by the injection of enormous quantities of blood into susceptible animals; and although for long periods recovered animals show no signs of the disease, they are subject to relapses, when once again parasites are found in the blood and plasmospheres resembling Koch's bodies in the internal organs. Again, *T. dispar*, (6) the cause of a theileriasis of cattle in Algeria, resembles *T. parva* in every respect, except that it can be transmitted by blood inoculation. Too much importance, therefore, should not be attached to the recognition of parthenogenesis. Is it not possible that, in spite of the failure to find parasites in the

recovered animal, they may be present in some unrecognised form which may remain latent or dormant until such time as the influence which restrains them is relaxed and their development can recommence ?

We are unable to suggest what form such a " latent " parasite might present or where it might be found. Gonder admits that " the first schizogonous generation may be repeated several times." He says, " with the formation of the gametocytes the cycle of *T. parva* in an animal arrives at a definite conclusion, and the animal either recovers or dies." Is it not possible that, in animals which recover, certain of the asexual organisms may survive and remain quiescent until circumstances arise favourable to the resumption of their vegetative reproduction ? Gonder himself, in describing the disappearance of the parasite from the organs of the recovering animal, says " the development of parasites in the body is arrested," although later he assumes that because the recovered animal is supposed not to relapse, " the parasite must perish." It is a well-known fact that the development of unicellular organisms may be arrested and resumed after longer or shorter intervals. Golder's theory appears to hinge upon the fact that parthenogenesis has not been recognised, but the idea that the parasite may be arrested indefinitely in the asexual stages disposes of the necessity for parthenogenesis.

Those who have had practical experience of East Coast fever are well aware that it frequently happens that certain animals show an elevation or irregularity of temperature, and even clinical manifestations of the disease, for a few days and then apparently recover. Sooner or later, some of these animals develop the disease in an acute form. The experience is so common that, in the " temperature-camp system " for the control of the disease, it became the rule to destroy at the first elevation of temperature. Of course the question arises, Were such cases due to recurrence or reinfection ? If the former, then arrested development of the parasite must have occurred ; if the latter, the theory that recovery results in immunity falls to the ground.

Watkins-Pitchford, in his report on " Dipping and Tick-Destroying Agents," Part 2, Schedule D, (7) shows the influence of dipping cattle in arsenical solutions in lengthening the period of incubation of East Coast fever from an average of from nine to at least sixteen days. The number of animals comprising his experiments, however, was small, and some of the cattle exposed failed to become infected. It is possible that a more exhaustive investigation might show that the development of the parasite in " dipped " animals is prolonged for a longer period than hitherto suspected.

In my Annual Report for the year 1919, (8) attention was drawn to certain circumstances in connection with outbreaks of East Coast

fever, which suggested that the regular dipping of infected cattle every three days in standard solutions of arsenic led to a suppression of the disease. This was indicated by (1) the large proportion of deaths among oxen as compared with other classes of cattle on certain infected farms during the winter, when tick life was practically dormant ; and (2) the recrudescence of the disease in many outbreaks where regular dipping had been practised as a method of control during those months when the rainfall was heaviest. The high mortality among oxen, as compared with cows and calves, could not be solely attributed to tick infestation. If a number of ticks, protected in the brush of the tail from the effects of the dip, were responsible, all classes of cattle would have been equally susceptible. It was suggested that the more rapid elimination of the residual arsenic from working animals, through sweating and urinating during exercise, caused a more rapid elimination of the residual arsenic, so that the development of the parasite arrested by the arsenic could be resumed. Here, again, the explanation may be that the development of the parasite is checked.

It is therefore certain that, in ordinary circumstances, the course of the disease may be arrested and that relapses occur. It has also been proved that the incubation period may be prolonged from nine to sixteen days as the result of spraying infected animals in arsenical solutions ; and it would appear that this delay may be indefinitely extended by systematic dipping in solutions of standard strength. If, therefore, the development of the parasite can be delayed for a week, a month, or a year, it is possible that it may be postponed for even longer periods ; and if such delay can be brought about by arsenic, it is probable that the production of immune bodies may exert a similar restraining influence. In piroplasmosis a tolerance is set up between the parasite and the host, as the result of natural recovery or treatment with trypan blue, but this breaks down when the animal is suffering from some other disease, such as rinderpest or East Coast fever. Or, again, in trypanosomiasis the disease may be arrested by appropriate drugs, or even under natural conditions, but relapse and even death occurs when tolerant animals are exposed to heavy rains, starvation and overwork. If *T. parva* persists in a latent form in recovered animals, the reappearance of East Coast fever in herds containing so-called " salted " animals after intervals of three, four, or five years might be attributed to the breaking down of resistance in recovered animals as the result of old age or adverse conditions.

A similar controversy has taken place in connection with the relapses of malaria, which are known to occur in malarial subjects

years after their departure from a malarious country to one where reinfection cannot occur. These were originally attributed by Grassi to parthenogenesis, and Schaudinn claimed to have demonstrated its occurrence in a case of benign tertian malaria before the first paroxysm of a relapse occurred. In the light of a recent criticism by Thomson (9) this theory cannot be accepted.

"Another view, associated with the names of Bignami, Ross, W. M. James, and others, assumes that schizogony goes on without intermission throughout the intervals between relapses. No essential difference in genesis is recognised between recurrences, where the intervals between recurrences approximate to the incubation period, and relapses which occur after long, healthy intervals. In the former case the interval may be prolonged by the continuance of special conditions, such as the exhibition of quinine. By a process of natural selection schizogony is kept up, but the number that survive at any given time are too small to produce symptoms or to be found in the peripheral blood. When the special conditions are removed, a relapse occurs. The resistance of the host, expressed in terms of antibodies, supplies a parallel explanation in the other cases. The human organism, in response to repeated attacks, is to be conceived as producing antibodies which resist the multiplication, as well as others which resist the pyrogenetic action of the parasites, and so long as a certain balance is maintained, it is quite conceivable that schizogony, to a limited extent, may be continued." This theory is practically identical with that submitted in connection with the recrudescences of East Coast fever.

"Another view is that the parasite may survive by finding its way out of the blood stream. Merozoites, it is thought, may penetrate into endothelial cells and, adapting themselves to altered nutritive conditions, enter upon a resting stage.

"It may be noted that S. P. James has recently (1917) made the suggestion that persistent or latent forms, which have hitherto escaped recognition because of the general resemblance to gametocytes, may be the cause of true relapses as distinct from recrudescences" (Thomson and Woodcock).

From the foregoing extracts, taken from one of the most recent works on protozoal diseases, it would appear that the same difficulty occurs in explaining the relapses of malaria as the suspected recrudescences of East Coast fever. The problem in both cases appears to be as to whether there is a latent form of the parasite, and if so, the nature of the parasite in its dormant condition. In neither case is a definite scientific proof forthcoming.

At the present time, owing to the recent outbreaks of East Coast

fever in Southern Rhodesia, the controversy has become very acute and of some considerable importance, because it is felt that if the "salted" animal can remain a source of danger the methods of controlling the disease will have to be modified. Indeed, at the time of writing, the slaughter of infected herds is being resorted to in place of the system of dealing with the disease by short-interval dipping.

It is to be regretted that the exponents of the two theories should tend to range themselves in separate camps: it were better if the so-called practical observers and those who profess and call themselves scientists could collaborate in an attempt to arrive at the truth. A recent writer on the subject has definitely stated "that in the very great majority of, if not in all instances, the cause" (of recrudescences) "has been due to a relapse of the disease in some salted carrier of the parasite, and that this has been proved as far as it is humanly possible to do so by field observations." This may be so, but it has not been proved scientifically. If the origin of the disease could be so clearly associated with certain recovered animals, it is strange that those so anxious to incriminate them should have neglected to obtain the only evidence upon which to do so, namely, the demonstration of the parasite in them.

It is only recently that suitable accommodation has been provided at the Salisbury Laboratory for research into this disease; hitherto it would have been too dangerous to keep infected animals under observation under such conditions that the danger of disseminating the disease could not be excluded. Now, however, that suitable provision is made the investigation of the problem can be scientifically carried out.

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SOME NOTES ON SURRA IN THE CAMEL, ITS TREATMENT AND PREVENTION.

By Major V. C. LECKIE, D.S.O., M.R.C.V.S.,
Royal Army Veterinary Corps.

GENERAL REMARKS.

A very considerable advance has been made in the treatment of surra during the past few years. The method introduced by Captain H. E. Cross, late Camel Specialist to the Government of India, viz. repeated intravenous injections of a solution of potassium antimony tartrate (tartar emetic), was in his hands most successful, but in the hands of less experienced people has at times produced a high mortality amongst the treated.

These notes are therefore put forward, giving the writer's two years' observation and experience of the treatment, in the hope that this may prove useful to those undertaking such treatment without former experience.

Further, the undoubted great value of sodium antimony tartrate in the treatment of surra in camels has not been previously recorded, and some experiments carried out at Lahore with this salt are added to these notes. In so far as "prevention" is concerned, it is thought that experience and observations made with Silladar Camel Corps may prove useful in dealing with the disease in other domestic animals and the handling of camel transport in general.

Surra for many years has been described under three headings, according to symptoms, viz.: (*a*) Acute, (*b*) Sub-Acute, and (*c*) Chronic. It is well known that many untreated camels die of surra while in the acute stage, and those that survive may put on condition and pass into the chronic stage, where little or nothing wrong may be noticed but trypanosomes, probably only few in number, being periodically found in their blood.

This may account for the fact that sometimes camels are found in excellent condition with but few trypanosomes in their blood whilst registering a low body temperature, and it is probable that under this heading the so-called "natural cures" may be placed, but it remains to be proved whether their blood is or is not infective. Zemindars (Indian agriculturists) who often keep camels, when they realise that their camels are sick from surra, put them aside and feed them generously for one or two years till the animals are fat and the disease has passed into the chronic stage (i.e. the animals have acquired naturally a resistance to the disease). Zemindars undoubtedly lose a good many in the acute stage of the disease, but

many pass from the acute stage to the chronic, and it is said that for two or three years the camel will have periodical attacks of the disease and eventually recover, that is, he may not become periodically ill from surra, although perhaps he may be a potential carrier.

It is thought that the stages of the disease may possibly be divided as follows :—

Acute.	Sub-acute.	Chronic.
Lasting three or four months up to as long as six months.	Beginning from four to six months after the first attack and lasting as long as two years.	Beginning in some cases a year after the first attack and lasting about three years.
Trypanosomes appear in the blood three or four times during a month.	Trypanosomes appear in the blood once or twice a month.	Trypanosomes appear in the blood once in six months or once in a year.
Most dangerous stage.	Not fit for any work.	Fit for work except during paroxysms of fever.

Symptoms.

For convenience sake the symptoms are grouped under two headings, acute and sub-acute being recorded together, as it is difficult to assert definitely that certain symptoms are found in acute and not in sub-acute cases, and vice versa.

Acute and Sub-Acute.—During the paroxysms the body temperature rises from 101° to 103° F.; the animal constantly lies down, stares steadily in front of him, refuses food and water; the coat looks harsh, and hair is very easily pulled out from the tail. Trypanosomes may be found in the peripheral circulation, varying in number according to the degree of the infection, or to the rapidity with which trypanosomes are multiplying. They increase in number as the temperature rises and decrease as it falls, till the normal is gained and the blood may appear microscopically negative.

The camel soon becomes emaciated, and muscle atrophy, particularly of the hump, back and loins, is noticeable; the hump in sinking may lean to one side, and is soft or flabby to the touch. Petechial hæmorrhages may often be found on the conjunctivæ and

nictitating membranes; serous swellings may sometimes be seen at the base of the neck and in the region of the chest pad, extending along the abdomen as far as the prepuce; opacity of the cornea and ulcer of the cornea may occur; the urine, which is normally very pale in colour, may be dark or even blood-tinged; swelling of the knee and hock joints may occur with eventual abscess formation. There is commonly abscess formation in the submaxillary, prepectoral, and inguinal glands, but abscesses may occur anywhere in the body.

Between the intervals of paroxysms of fever which occur concurrently with the increase in number of trypanosomes in the peripheral circulation, the animal may feed well, but as the disease progresses it becomes weaker, more emaciated and dejected in appearance. Therefore many camels suffering from surra in the acute and sub-acute stages are often put down by the inexperienced as cases of debility, requiring only rest and good feeding.

Only certain of the foregoing symptoms may be exhibited in a given case, but a systematic "blood test" of the suspected will in a very high percentage of cases reveal trypanosomes in the blood. The under-fed or overworked camel will not present periodical rises of temperature and will not be continually going off feed. As a rule it will eat ravenously and will respond quickly to rest and good feeding.

Brain symptoms are not an uncommon occurrence in the acute and sub-acute stages. The camel becomes very restless, breathing is accelerated, it froths at the mouth, and if tied down it will struggle violently; when free it will gallop till it falls exhausted, death generally supervening.

In the early stages of the disease the lungs are sometimes affected. This condition is difficult to distinguish from the so-called "influenza" of camels, but it does not spread from camel to camel. The condition appears to be of the nature of a broncho-pneumonia. The camel constantly lies down; from time to time it lowers his head almost to the ground, and a long-drawn mucous "râle" is heard. This is in the nature of a cough in other animals, but there is much less expiratory effort, and the circumstance might pass unnoticed but for the fact that it is repeated at intervals. Post-mortem on such camels generally reveals pneumonia of both lungs, and if the condition has been in existence for some considerable time, large necrotic and gangrenous areas may be found.

The cessation of rumination is perhaps one of the earliest symptoms presented. The Sarwan (camel man) having noticed that his camel has browsed indifferently during the day, further notices in the evening that it does not ruminate. He is, however, apathetic on

the subject of surra ; he knows his camel to be infected, but keeps the information to himself, preferring to work the animal for as long as it will live, applying such method of treatment as he himself understands, e.g. thermocautery applied to the chest walls and sub-lumbar regions, or drenching the animal with an admixture of its own blood in milk or water.

Chronic.—The appearance of the animal will alter considerably under this heading, according to whether the disease progresses favourably or unfavourably to the subject. If the camel is gaining a natural resistance to the disease, it may have no visible indication of any indisposition ; it may appear to all intents and purposes in perfect health—fat, muscular, coat sleek, eye bright—yet at long intervals trypanosomes may be found in its peripheral circulation, generally few in number, and remaining only for a few days. This may indicate that the camel has been well cared for and not overworked while in the acute and sub-acute stages. If the disease is progressing unfavourably to the subject, it will remain debilitated, still presenting muscle atrophy and often chronic thickening of the prepectoral, submaxillary, and inguinal glands, and sometimes having a chronic chest pad injury. It will be hide-bound, tucked up, with harsh coat, and the hump for the most part gone.

Mode of Transmission of Surra.

It would appear in the present stage of our knowledge of the disease that the transmission of surra from camel to camel or from some other "reservoir" to camel (e.g. buffalo, ox, horse, etc.), is generally a mechanical act, and that the majority of the known agents implicated in the transmission appear to do so in a purely mechanical way. Nothing is yet known of a life cycle of the trypanosomes taking place in the body of such agents, except in the case of a tick, *Ornithodorus Crossi* of the family Argasidæ, which Cross proved was capable of transmitting surra as long as a hundred and one days after feeding on an infected animal.* But much investigation is still required in this direction with regard to other vectors.

Amongst the many agents involved in the spread of surra are primarily the biting or blood-sucking flies, also ticks, lice, fleas, and birds. Several species of the family Tabanus have been experimentally proved to be the transmitters of the disease. The Stomoxyidæ have been suspected, but nothing definite has as yet been proved against them in so far as the disease in the camel is concerned. The

* Department of Agriculture, Punjab, Veterinary Bulletin No. 6 of 1921.

stomoxys when examined after feeding on a surra-positive camel will be found to contain live trypanosomes in its engorged stomach. It is also to be seen drinking from the drop of blood which wells up after Tabanidæ have taken their fill. Moreover Stomoxyidæ, at certain times of the year, are to be seen in hundreds covering fresh wounds, palan galls (caused by camel pack-saddles), etc., of surra-infected and non-infected camels, so that they are worth serious consideration as being possible vectors of the disease. But it is significant that if precautions are taken to keep camels away from areas infested by tabanus, surra is very materially reduced. Surra is principally spread from camel to camel, they themselves acting as their own reservoirs. Other animals that they come into close contact with in villages, viz. the ox and the buffalo, are carriers of the disease in a benign way, i.e. do not carry a sufficient number of trypanosomes in their blood to be a danger; but if these animals were responsible for conveying the disease to camels, hardly a camel in the Punjab could escape. Captain Cross has said that 25 per cent. of the camels in the Punjab are surra infected, and this has been approximately the figure found in dealing with surra in Silladar Corps in the Lahore district.

It is not proposed here to go into the life history of tabanus, or the so-called surra fly, the principal agent in the spread of surra in camels, for it has been excellently recorded in details elsewhere.*

It will suffice to say that several species have been experimentally proved to be transmitters of the disease, and that only the female fly is involved. An interrupted feed is necessary, that is to say a female fly feeding on the blood of a surra-positive camel may be interrupted before it is completely engorged, either by the animal lifting a foot suddenly, whisking his tail, or swinging the head round. Being disturbed, she may alight on another camel browsing near by, this time, let it be supposed, surra free; the piercing and drinking organs are now smeared with the blood of the first camel, and therefore it may be assumed contain trypanosomes, and in finishing her feed on the second camel she has been the means of conveying surra from the first. One might well imagine the possibility of this act being continued until the fly became engorged. On the other hand, should she complete her meal undisturbed (on one camel), she will fly away and digest her food, will not feed again for some hours, and as far as is known at present is then incapable of communicating surra as a result of the last feed. Further investigation is, however, required as to the possibility of the fly becoming infective again after a period,

* Department of Agriculture, Punjab, Veterinary Bulletin No. 5 of 1921. Cross and Patel.

i.e. a life cycle taking place within the fly's body, as is the case with tsetse fly in sleeping sickness. Tabanus appears in the Punjab plains from about the 10th to the 15th of March, and is to be found till November 15th or 20th, and in the hills and foot-hills from about April 1st to October 31st. There is a superstition amongst Punjabi camel men that the fly in the plains makes its appearance when a certain small green shrub, known as "harmal," breaks through the ground. This is probably only coincidental, but it is of interest in so far as the camel men take their camels away from fly areas as soon as it is noticed.

Of other possible vectors of the disease, the camel louse has fallen under suspicion, and the engorged stomachs of lice taken from a surra-positive camel have been found to contain live trypanosomes. Certain experiments were carried out at Lahore to see if it was possible to communicate the disease to guinea-pigs, by allowing such lice after periods of starvation to feed on healthy subjects. The guinea-pigs used did not present trypanosomes in their blood.

With regard to fleas it was conjectured that they were the means of spreading surra in an outbreak which occurred in 1923 amongst foxhounds at Lahore. After the original cases were isolated, additional cases cropped up at intervals, but when the kennels were thoroughly disinfected and precautions taken to exterminate the fleas, no further cases occurred. In former years the disease had spread through the pack, necessitating its complete extermination. Leese mentions in his *Tips on Camels* that whole populations of village dogs that have fed on the carcases of surra camels are wiped out, and such has been the experience at Lahore. The jackal feeding on carcases in like manner was possibly the origin of the outbreak amongst the above-mentioned hounds. However, Cross's transmission experiments with fleas appear to have been negative.

Of other mechanical agencies, birds should receive consideration, for the ubiquitous Indian crow is very frequently seen pecking at a fresh wound, chest pad injury, or palan gall. But the number of cases so produced must be small and only needs notice in passing.

During outbreaks of camel pox amongst young stock, the sarwan is reputed to take a thorn and go from one camel to another scarrifying and puncturing as a rough and ready method of vaccination, and as the same thorn may be used until it breaks, the possibility of spreading surra in this way may be readily understood.

But a possible method of "spread" of more practical importance is that of the careless takers of blood-smears and the omission to thoroughly sterilise a needle between the piercing of the ear of every camel submitted to blood-test. It is therefore necessary to issue

the most definite orders with regard to smear-taking. Only authorised persons who realise the danger of spreading the disease by this means should carry out this work.

TREATMENT.

The treatment may be briefly stated as an attempt to destroy the trypanosomes *in vitro* by gradually saturating the system with potassium antimony tartrate, the method employed being to introduce intravenously, at body temperature, certain quantities of a 1 per cent. solution of potassium antimony tartrate (tartar emetic), the solution being made by dissolving 1 gramme of the salt in a 100 c.c. of boiled, distilled water, the whole being filtered before introduction.

Reference will be made in these notes to treatment by sodium antimony tartrate, but as the method of preparation of the solution and the technique in administration for this salt of antimony is identical with that employed in treatment by potassium antimony tartrate, it is proposed only to describe in detail treatment with the potassium salt.

The Apparatus.

For purposes of description the apparatus is best divided into four sections: (*a*) for the preparation of solutions for injection, (*b*) for the preparation of the subject for injection, (*c*) the apparatus required for the administration of the solution, (*d*) microscopical.

(*a*) Apparatus Required for the Preparation of Solutions for Injection.

1. *Glass (graduated) urine flasks*, 2,000 *c.c.* Required for measuring the daily quantity of distilled water for making up the tartar emetic solution and physiological salt solution.

2. *An accurate scale and set of gramme weights.* For measuring out accurately amount of potassium antimony tartrate and physiological salt as above.

3. *Enamel jug and basin (large).* For dissolving the salts respectively as above in boiling distilled water; glass is unsatisfactory.

4. *Glass funnels (large size), filter paper sheets (large), irrigation douche (enamel), fitted with india-rubber tubing and fine glass nozzle with clip.* For filtering solutions.

5. *Erlermeyer flasks (glass)*, 1,000 *c.c., a "Hearson" biological incubator.* For daily stock solutions. These when filled are kept in an incubator at body temperature till required. If it is required to

make one's own distilled water, a copper still would be required in addition.

(*b*) Apparatus Required to Prepare the Camel for Intravenous Injection.

1. *Large curved scissors, aseptic razor, metal iodine swabber.* For preparation of the site for intravenous injection.

2. *Two ropes* 15 *feet, two ropes* 4 *feet.*—For securing and fixing the fore and hind limbs, and for additional control of fore limbs.

(*c*) Apparatus Required for the Actual Introduction of the Solutions.

1. *A glass flask* (*Erlich's salvarsan*). A convenient shape of flask; as the mouth is wide it makes the pouring in of solutions easy.

2. *The above fitted with* 2 *feet* 6 *inches of india-rubber tubing to the end of which is attached a* (*shortened*) *intravenous injection needle.* A window in the form of a 2-inch glass tube may or may not be fitted 3 inches from the needle as found to be necessary (see text).

3. *A metal spring clip.* The spring clip is placed on the tubing as close as possible to the needle.

4. *Three Erleymeyer glass flasks* 250 *c.c. each.* For the actual dose measured for each camel, labelled, " salt," " T.E." and " salt."

5. *Graduated glass flask* 200 *c.c.* For measuring out doses as above from the stock solutions in the incubator.

6. *Methyl alcohol.* For sterilisation of injection needles between injections.

7. *An oil stone.* For sharpening injection needles.

(*d*) Microscopical Apparatus.

1. *Microscope glass slides, covered glasses.* For diagnostic purposes. Frequently in use at the commencement of and constantly throughout the treatment.

2. *Spirit lamp, stains, microscopical outfit.* For smear work.

The preparation of the solutions is best understood by reference to an example of a daily injection sheet as used at Lahore. This sheet gives the individual camel numbers, record of temperature on that date, and notes against certain camels, such as urine discoloured, constipation, off feed, etc., and whether or not an injection is to be given: if to receive an injection, the dose of 1 per cent. solution of tartar emetic and physiological salt to be stated.

The whole of the tartar emetic solution and physiological salt solution is calculated and recorded at the foot of this sheet. Having

weighed out the required amounts of physiological salt and potassium antimony tartrate, the respective amounts of distilled water are measured in the glass urine flask capable of containing 2,000 c.c. The distilled water is then boiled and the salts are dissolved in their respective quantities of boiled distilled water; enamel basins are best for this purpose. The solutions are then filtered, and the filtered solutions poured into their respective stock flasks, which are labelled or marked, and put into the incubator till required.

TABLE I.

DAILY STATEMENT OF INTRAVENOUS INJECTION.

Camel No.	Temperature.	Remarks.	Dose T.E. c.c. 1 %	Dose Saline c.c. 1 %	Notes.
54th C.T.C.					
175	97°		50	50	
40	98°		100	50	
658	97°		175	50	
54	99°		50	50	
158	99°	Urine discoloured	No injection		Mag. sulph., 2 lb.
754	97·2°		100	50	
445	98·4°	Urine discoloured	No injection		Mag. sulph., 2 lb.
658	98°		100	50	
371	103°	Constipated	No injection		Mag. sulph., 4 lb.
429	98·4°		100	50	
581	99°	Mucous membrane yellow	No injection		Mag. sulph., 2 lb.
424	97°	Mucous membrane yellow	No injection		Mag. sulph., 2 lb.
762	98°		100	50	
223	100°	Off feed and constipation	No injection		Mag. sulph., 4 lb.
656	98°		200	50	
670	98°		150	50	
197	98°	For blood examination	50	50	Sick fourteen days
155	97°		100	50	
665	97°		175	50	
336	101°	Constipated	No injection		Mag. sulph., 2 lb.
		Total	1,450	650 c.c.	
		Total	14·50	6·50 gs.	

The day stock solution is 14·5 grammes tartar emetic in 1,450 c.c. distilled water, and the day's stock solution of physiological salt solution is 6·50 grammes of physiological salt in 650 c.c. of distilled water,

Those camels which are to receive an injection are then paraded and prepared for injection—six at a time. They are "trussed" in the sitting position, four ropes being required for the process, as follows :—

1. A rope is passed over the base of the neck and under each forearm (from the inside), the free ends are then brought back and fastened over the base of the neck. An additional short rope on each forearm, including the pastern joint, completes the "fixation" of the four limbs. Care must be taken that a forefoot does not exert any pressure on the jugular vein to be used, as this will effectually stop the flow of the solution into the vein.

2. The hind limbs are fastened by passing a rope over the loins, inside the stifles, and round what corresponds to the "pastern" in

FIG. 1.—A Camel roped and fixed ready to be turned over for Intravenous Injection.

the camel, the free ends being brought up and fastened over the loins behind the hump.

The camel, by reason of his peculiar conformation, is easily rendered immobile in this manner. The next stage of procedure is to turn the animal over on his side. This is done by the sarwan standing at the head, grasping the lips in both hands, bending the head back towards the hump, while three other sarwans push at the shoulder, hump and quarters, reminding one of the first line of a Rugby scrimmage. The process of turning over is carried out to the accompaniment of a good deal of noise, constant complaint from the camel, and shouting on the part of the sarwans.

The sarwan at the camel's head still retains his hold of the animal's lips after it has been turned over. After a camel has been thus dealt with once or twice, he becomes very cunning, and does all he can to prevent it happening again ; he does this by turning out his stifle as far as he can on the opposite side to which he is being pushed, and no amount of exertion on the part of the camel men

will turn him over till he is made to put his stifle back in position again.

It may be assumed that the injection site over the jugular vein has already been clipped, shaved, and cleansed, and that the camel is ready to receive his injection. Reference to the daily injection sheet gives the individual dose to be intravenously injected.

Two small glass flasks are taken and into each is poured from physiological salt solution daily stock in the incubator 25 c.c. of solution. These are marked with a wax pencil "salt": they are to be used immediately preceding and following the dose of tartar emetic.

The required dose of tartar emetic is then poured into a third glass flask from the stock solution in the incubator, and is marked "T.E.", so that we have the dose in three flasks in the order in which they are to be used, viz. (*a*) salt, (*b*) tartar emetic, and (*c*) salt.

FIG. 2.—Showing the Jugular Vein standing out prominently behind the Angle of the Jaw with the Needle Apparatus in Position.

THE INTRAVENOUS INJECTION APPARATUS.

This requires little description. It consists of a cylindrical, graduated glass flask, fitted with an india-rubber tube 2 feet 6 inches long, to the end of which is fitted a shortened intravenous injection needle. Three inches from the needle, 2 inches of glass tubing is let in as a "window," and as close to the needle as possible the tube is fitted with a small metal spring clip.

THE TECHNIQUE EMPLOYED IN THE ADMINISTRATION OF THE SOLUTIONS.

Having been prepared for injection, the head of the camel is moved backwards or forwards till in such a position that the jugular vein exposed stands out like a garden hose pipe. An assistant holds the apparatus by the glass flask and the operator pours into the flask the contents of the first small dose flask marked "salt" (25 c.c. normal saline). He then takes the needle of the apparatus, releases

the clip, and raising and lowering the needle, allows a small quantity of the saline solution to escape through the needle, thus ensuring that there is no bubble of air that may act as a " block." He then refastens the clip.

He now takes the needle between the thumb and forefinger of his right hand and, bearing his weight on his right leg, leans down and points the needle to the jugular vein at its greatest prominence, which is about 3 or 4 inches from the angle of the lower jaw. In this position, with his arm slightly bent, he gives a short, sharp thrust, and the needle passes into the jugular vein. An alternative method is to keep the arm quite straight from the hand to the shoulder, touching the jugular with the needle point in the same manner and the bearing down with one's weight gradually till the needle pierces skin and jugular. This is probably the simplest method.

The clip is now released and the normal saline in the flask of the apparatus begins to descend. Before it has completely disappeared, the operator pours in the contents of the dose flask containing the tartar emetic solution marked " T.E.", and again before this is quite descended into the tube, the contents of the second flask marked " salt " is poured in. As this disappears into the tube, the needle is smartly whipped out, and a metal swabber dipped in tincture of iodine applied to the puncture made by the needle. The operation is now complete, and the camel may be unroped and allowed to rise.

So far it will be noticed that no mention has been made of the " glass window " in the rubber tubing of the apparatus, because in the hands of an expert in the use of the apparatus it is unnecessary, and its only *raison d'être* is when there is some uncertainty as to whether the needle has properly pierced the jugular vein. If there is any doubt about this, the glass flask of the apparatus should be lowered, and if the lumen of the needle is cleanly put into the jugular, blood will be seen to pass into the glass window. In that case the injection may be proceeded with, but should the blood not flow into the glass window, there is either an " air block," the needle is clogged, or it has not properly punctured the jugular. In this case the needle must be at once withdrawn and the operation recommenced.

From the foregoing, the value of the two doses of saline solution may be understood. Tartar emetic solution, if allowed to escape between the skin and the jugular, causes most serious swelling and sloughing. Therefore saline is employed to ensure a clear passage into the vein for the tartar emetic solution, and finally to wash out all tartar emetic solution from the lumen of the tube and needle before the needle is withdrawn.

(*To be continued.*)

"HÆMOGREGARINA CANIS."*

By M. ANANT NARAYAN RAU, G.M.V.C.

This parasite of the dog was first discovered by Bently in 1905 in the peripheral blood of dogs in Assam. James described it in the *Scientific Memoirs* by the Officers of the Medical and Sanitary Departments of the Government of India, No. 14, of 1905. Christophers published the mode of the schizogony of the parasite in the bone marrow alone (*Scientific Memoirs*, No. 26, 1906), and also published the sporogony found in the common dog-tick, *Ricinus sanguineus* (*Scientific Memoirs*, No. 28, 1907). There have been to my knowledge no veterinarians or medical men in India, other than those mentioned above, to have worked further either to confirm what Christophers wrote or to add anything new regarding the parasite in question. In all the books on veterinary subjects to which I have had access, no mention is made whether *Hæmogregarina canis* is pathogenic to dogs or not. James and Christophers are the only two who stated that this parasite is non-pathogenic to dogs. This part of the question is a very important point to the veterinary clinician and will be discussed later. On the 2nd of December, 1921, this parasite came under my observation in the dog ward here, for the first time, in an imported dog (see Fig. 1) brought from Mesopotamia by a demobilised soldier. The history of the case was that it was all right till it was brought to Madras, and some time after its arrival it got ill. It is perhaps likely that the dog contracted the disease in Madras. Up till now, this parasite was reported only from Assam and Madras, so it is likely that the dog contracted the disease here. This dog gave an opportunity to study this disease, and the owner very kindly left the dog as an in-patient for over a month. It was discharged from the hospital, and was under observation till the 2nd of February, 1922, when it died.

In May 1922 these parasites were seen in the smears of blood of a dog sent from Cocanada Veterinary Hospital, and also in those of a dog at Saidapet Veterinary Hospital. Engorged ticks were obtained from the former dog, and the latter was obtained through the very kind help of Mr. A. J. Wilson, G.M.V.C., to whom my thanks are due. These three cases and six experimental dogs gave enough material for microscopical study of the schizogony, sporogony, and the clinical symptoms of the disease.

In the latter half of 1923, blood smears from over two hundred neglected puppies and young dogs from various parts of Madras

* *A paper read at the Third All-India Veterinary Conference, Madras, December* 30, 1924.

showed only about 7 per cent. of them to harbour these parasites, of which six showed light infection. In the Madras Veterinary College, smears from dogs from the wards and also from the moffusil are examined for one thing or another, but as far as I could gather no smear has been reported to have contained *H. canis* excepting in those three cases mentioned above, when I had the good fortune to detect the parasite in them. This fact shows that this disease is much less common in well-cared-for dogs than in the neglected pariah dogs. This point is an important one, and will be taken up again while discussing the mode of transmission of the disease. The blood smears of the dog from Saidapet showed a very scanty infection in the early stages, and Christophers is right in stating that scanty infection might lead one to suppose that the dog is free from infection.

Description of the Parasite in Peripheral Blood.

It is an elliptical or an oblong parasite usually seen in a leucocyte which looks like a polymorphomononuclear cell (Plate I, *A*). Sometimes it is found in a transitional cell also. The host cell forms a "cytocyst" for the parasite. In a smear that is well stained by Giemsa's stain, a very thin pink zone is seen to line the cytocyst. Sometimes in the blood smears one encounters a few of these bodies free, perhaps due to the leucocytes being ruptured while making a smear. In such bodies also one can see the pink stained zone forming the boundary of the oblong body. This is apparently a capsule of the parasite derived from its own protoplasm. Sometimes the parasite leaves a space between itself and the capsule, which is at times very marked on either side of the middle of the parasite. Even in the free forms this is clearly seen. This suggests that the parasite has vermicular movements within the capsule, hence this appearance. Vermicular movements of the parasite in fresh blood cannot be seen owing to the refractile character of the capsule which hides the contained parasite. In a stained specimen the nucleus of the parasite is seen generally at one end of the ellipsoid body, like one of the centres of an ellipse. The nucleus may be round or oval, compact or loose or even **U**-shaped. It is not uncommon to find the nucleus at the middle, where it may be rounded, elongated, or even **U**-shaped, but in such a situation the nucleus is invariably reticulated with clear unstained specks in it. In a well-stained blood smear one can see a few pink stained granules strewn in the space within the capsule of the oblong parasite. Perhaps these granules are chromidia or extra nuclear chromatin. At first one may think that this stippling may have been only in the cytocyst, but it is seen even in free parasites

mentioned above. The body of the parasite, though inside the cell host, does not seem to touch the nucleus of the latter. Very often a strip of the host's nucleus is seen stretching across the diameter of the parasite. The cell containing this parasite does not seem to be bigger than a normal one, and Christopher says that the presence of these parasites in the protoplasm cripples it, because its movements lack the vigour of the healthy leucocyte, and that it scarcely moves from its original position when observed under the microscope. It is usual to find only one parasite in a cell, but two are also met with. Generally there is only one nucleus in a parasite, but it is not unusual to find two nuclei either close to one another or at opposite ends of the parasite. There is no periodicity in their appearance in the peripheral blood as is found in *Microfilaria recondita*, Grassi (*Agricultural Bulletin*, No. 144, by M. Anant Narayan Rau, 1923).

In stained blood smears one encounters degenerating parasites, which stain very faintly, hence the conclusion that they are either partly or completely dead ones.

Description of the Parasite.

(*In citrated blood as a hang drop or examined between a slide and coverslip.*)

In fresh blood examined under a microscope the parasite looks like a refractile oblong body in the host cell, and the structure of the parasite cannot be made out. Within about an hour or so after drawing the blood out of the dog, elongated parasites, looking like bananas, emerge out of their cytocysts. These parasites or "vermicules" have been observed actually coming out of their cytocysts. The removal of the blood from the body of the dog seems to be enough stimulus for the parasite to become free vermicules. Such vermicules are also easily obtained from the gut of engorged ticks. This proves the fact that the parasites found in the peripheral blood are "sporonts." The sporont when observed in fresh citrated blood shows, after a little while, a distinct bulge of the capsule beyond the contour of the host cell. This is apparently caused by the movement of the enclosed parasite. The capsule bursts, and one can then see the pointed end of a vermicule protruding out of the opening formed, and bending first to one side and then another as if to widen the aperture for it to escape. After the lapse of about fifteen to twenty minutes nearly half of the vermicule comes out, when a sort of peristaltic movements may be observed in it. These movements help the vermicule to crawl out altogether in about thirty to forty minutes. It is difficult to say if the parasite rests doubled up in the capsule

so as to cause the nucleus to appear situated at one end of the oblong sporonts. Christophers says that when the parasite is first liberated it is stumpy in form, differing from the encapsulated form only in not possessing a capsule. Observations made here leads one to confirm this statement. The newly liberated vermicule is stumpy, but is a little longer and narrower than a sporont, and at the same time it shows a few vacuoles in its protoplasm and has a slightly pointed end, which is an important feature. Once it is out as a vermicule it elongates rapidly, and its movements such as bending and straightening are well marked. A slow rotary movement is also seen sometimes, and a combination of all these movements help the vermicule to change its position on the slide under the microscope. A vermicule is pointed at one end and rounded at the other, and its nucleus is situated at its middle. (See Plate I, *C*.) The nucleus stains intense magenta with Leishman's stain, and it is compact in structure, though it looks a little bigger than that found in a stained sporont. Often it projects on either side a little beyond the contour of the vermicule itself. The protoplasm stains sky blue, and in the posterior half a few vacuoles are seen. Sometimes one or two vacuoles may be seen towards the pointed end also. A vermicule sometimes contracts to such an extent as to cause the distribution of its protoplasm round its nucleus as to form a round or globular body, yet the point of the acute extremity can still be seen projecting beyond its contour. It again resumes its original shape after a short time. Similar forms are seen plentifully in the gut of a recently engorged tick. This movement perhaps helps the vermicule to get into the intestinal epithelium of the tick. In a hang drop or in a drop of fresh blood between a coverslip and a slide, the liberated vermicules showed a tendency to agglomerate when kept for some time, even in smears made from such hang drops groups of these have been seen. They generally place themselves side by side in such groups. Why such a thing happens is not understood. Vermicules have been kept living for a period of forty-eight hours in hang drops kept at room temperature and up to seventy-two hours in an ice chest, and the only change noticed in them during this period was in the nucleus, which seemed to get loose in structure. At this stage one could compare it to a loosely entangled skein of twine.

Schizogony of the Parasite.

Christophers has described the schizogony of this parasite as occurring only in the bone marrow. Mauson, in his book on *Tropical Medicine* (1921), mentions the spleen also as a place where schizogony

takes place. So far no one seems to have shown the schizogony of this parasite of the dog taking place in the liver. I take this opportunity of recording that the encysted stages of *H. canis* are found in fairly large numbers in the liver also. In all the dogs that harboured these parasites the constant places wherein the schizogony took place were the bone marrow, the spleen, and the liver.

In the spleen, in addition to sporonts we find many other forms as well. In stained smears of the spleen-pulp after the death of the animal, or of the spleen puncture fluid smears taken when the animal was alive, one encounters a peculiar form of the parasite. It almost fills the protoplasm of the host cell, which in this case is a large mononuclear. These forms are also seen, sometimes free. (See Plate I, *D*.) They are round or oval bodies, whose protoplasm contains a few vacuoles. With Leishman's stain the protoplasm stains a sky blue colour, and the nucleus, which is situated in the middle, and which is composed of thirty to forty small rods, stains a deep pink. This form of the parasite is a schizont. Schizonts are seen plentifully in the bone marrow, and to a less extent in the liver also. James describes such forms as naked forms of *H. canis*. A schizont enlarges by stages to form a brood cyst in the large mononuclear leucocyte. It derives a thin capsule perhaps from its own protoplasm. As this cyst grows, the nucleus of the host cell is thrust aside and sporoblastic buds appear on the capsule of the cyst. At this stage the cyst refuses to stain and looks morular. (See Plate I, *E*.) In fresh spleen punctures material this is seen like a mulberry, and when such bodies are pressed between a coverslip and slide, from some of them, merozoites are squeezed out. These markedly resemble vermicules in their shapes and movements, but they are much smaller than the latter, and do not contain vacuoles in their protoplasm at this stage. In stained spleen pulp smears the number of these brood cysts can be seen to vary according to the severity of the infection. In serial sections of the spleen tissue one can easily study the interior of these brood cysts, and this helps to reveal the various stages of the schizont during its schizogony. The rod-shaped bits of nuclear matter of a schizont are seen to travel towards and are arranged at the periphery of the globular brood cyst. These bits of nuclear matter ultimately form the nuclei of newly forming merozoites. (See Plate II, *A* and *C*.) It is remarkable to find thirty to forty merozoites in a cyst corresponding to the same number of rod-shaped granules in the schizont.

The merozoites have no capsules and are arranged beautifully in the periphery of the section of the cyst with the granules of the debris of the used up or residual protoplasm in the middle. (See Plate II, *A*.)

It is important to remember that a merozoite is naked and its central nucleus is compact. In some merozoites a few vacuoles develop in the protoplasm, and at the same time the compact nuclei also become loose in structure and later on break up into bits. Each of such merozoite is a newly forming schizont, which is taken up by the mononuclear leucocyte, and in it the schizogony is again completed. The merozoites with compact nuclei grow rapidly to form elliptical plumpy forms, broader at the posterior ends. The anterior ends have their protoplasm reduced in quantity, perhaps due to its flow towards the other ends. The effect of this flow is to make the nucleus appear at one end of the parasite. These forms are found in the transitional or polymorphomononuclear leucocytes, where they remain as newly formed sporonts, and get circulated in blood, so that they get a chance of being ingested by an intermediate host for the completion of its sporogony. Those schizonts which have exhausted their power of a sexual cycle evidently give rise to the latter forms of merozoites, which form the sporonts. These merozoites, when they are taken up by the leucocytes, protect themselves against the action of these cells, and vice versa, leading to the formation of a capsule and cytocyst, and enables the newly formed sporont to live in it till the life-time of that white cell, and then it may be ingested by another polymorphomous nuclear cell and so on.

In liver puncture smears or smears taken from scraping the section of the liver, during an autopsy, one sees schizonts and a very few merozoites, though not so many as are seen in those from the spleen or bone marrow. The presence of these led me to believe that there may be the completion of schizogony in the liver also. Sections of the liver did reveal cysts with merozoites in many of them. The number of schizonts in the liver being small, one should expect to find a correspondingly less number of cysts containing merozoites. (See Plate II, *C*.)

Sporogony of "H. Canis" in "R. Sanguineus."

The sporogony of *H. canis* was observed by Christophers in 1907 in the female tick *R. sanguineus*, and as far as I know he is the only one that has published his work (*Scientific Memoirs*, No. 28). I feel glad to take this opportunity of confirming much of his work. The material obtained for study consisted of ticks obtained from the three cases which were naturally infected, and also of home-bred ticks fed on one of the naturally infected dogs and three experimental dogs.

The stages of the developmental forms of *H. canis* are all obtained

mixed up with the contents and the epithelial cells lining the gut wall of the tick. With the aid of the home-bred ticks it was not very difficult to determine, more or less, the time taken by the sporonts, by stages, to give rise to sporozoites. In the gut contents of the ticks one finds a few leucocytes with sporonts in them, a few free sporonts, vermicules in various stages of development, and a few sporozoites. In the epithelial cells that are found free in the gut contents obtained after squashing a tick, one encounters vermicules dividing into daughter vermicules. One could even observe easily the conjugation of two of these daughter vermicules. These bodies that conjugate are sometimes of the same size, and sometimes one is found to be more slender than the other. Perhaps one can call the former a microgamete and the latter a macrogamete. Zygotes in various stages of development are also seen in the epithelial cells of the gut of the tick.

The home-bred ticks were let on a fairly heavily infected dog, and one or two of these were removed at intervals for study. Six hours after a tick had started to engorge itself, though the quantity of blood engorged by the tick is very small, yet a few vermicules were seen. Some of the sporonts were still in their capsules in the leucocytes, the latter showing signs of degeneration. In twelve hours most of the vermicules had been liberated. Some sporonts were seen free in the gut contents. Some showed signs of degeneration. The proportion of white cells to the number of vermicules is small, which means degeneration and disintegration of the host leucocytes. The vermicules looked exactly like those obtained from the blood *in vitro*. Their movements were not different in any way excepting that they seemed to assume the rounded appearance described above. How they actually got into the epithelial cell of the gut of the tick has not been observed. It is presumed that the straightening movement of the rounded vermicule enables it to force itself into the protoplasm of the gut cell. In twenty-four hours most of these vermicules were seen in the gut cells, especially those cells that were free from digestive granules, and they took up their abode far from the nuclei of these cells. The vermicule at this situation was more or less rounded in shape or even bean-shaped. (See Plate II, *E*.) The vacuoles in its protoplasm seemed bigger than in the previous stages, and at the same time the nucleus broadened itself loose and looked like a skein of entangled twine. In thirty-six hours the nucleus was seen to divide into two portions, in some the division was unequal; very often the cause of the two newly formed segments appeared to be a vacuole which enlarged and rent the vermicule, so to say, into two pieces longitudinally. At this stage fine chromatin granules appeared in the protoplasm at one or at both the ends of the daughter

vermicules. Sometimes this chromatin dust was seen strewn round their periphery also. This is the stage where sexual differentiation is completed and conjugation takes place only among them. Sometimes, though very rarely, four daughter vermicules may be seen in the same epithelial cell. Christophers has also seen this, and he states that he even saw eight of the daughter vermicules in one cell. (*Scientific Memoirs*, No. 28.)

The secondary vermicules are much more slender than the original vermicules. These are found free in the gut contents. They are perhaps discharged into the lumen of the gut by the bursting of the epithelial cell owing to their development. This accounts for the reason of the vermicule to station itself more at the margin of the cell away from its nucleus. When compared among themselves, some of the secondary vermicules are slender, but the majority of them are of the same breadth more or less. It may be that the former are microgametes and the latter are macrogametes. Conjugation has been observed to take place in the gut contents between a slender and a slightly stouter form. (See Plate II, *E*.) These place themselves side by side, fuse and form rounded or slightly bean-shaped bodies in about an hour after they had approximated. This has been observed under the microscope. The body thus formed may be called an " ookinet," and, strange to say, it does not contain the fine chromatin granules found in the secondary vermicules. The ookinet gets into a young cell of the gut, where it forms a more or less rounded " oocyst." These oocysts are found plentifully within forty-eight hours after the beginning of engorgement by a tick.

In the oocyst there are no vacuoles and the chromatin is loosely arranged at the periphery. The oocyst has a thin capsule. The nuclear substance breaks up into bits and gets arranged like knots in a net. At the same time the quantity of the protoplasm gets increased and sporoblastic buds appear on the capsule. These buds are not so prominent as those found in a brood cyst during schizogony, and are also more transparent than the latter. Each nuclear bit forms the nucleus of a sporozoite, and by apportioning the required quantity of protoplasm it forms its body.

These oocysts or sporocysts are situated very near the lumen of the gut, and the object of placing themselves is to discharge the sporozoites when the sporocysts burst open. In sections of these sporocysts the sporozoites are seen arranged exactly in the same manner as merozoites in a brood capsule, but the number varies from twelve to twenty. (See Plate II, *F*.) The sporogony is seen to be completed in about three days.

The sporozoites are small, though they are shaped like vermicules.

The nucleus of each is compact and more or less rounded. A few small vacuoles are seen in the posterior part of the body protoplasm. The movements of the sporozoites are exactly like those of vermicules, but are less active.

Mode of Infection.

Three young pups and three adult dogs were used for experiments. The pups were from a known bitch and were free from infection. The adult dogs were obtained from the lethal chambers and were kept under observation for over a fortnight. From the infected dog obtained from Saidapet, ticks were collected, and one of the adult dogs was infected with an emulsion of these. On the sixth day after injection the dog showed a rise in temperature, which continued for some days. On the fourteenth day after injection a very few parasites were seen in the peripheral blood of the dog. From the nineteenth day onwards the parasites were found to be numerous, and the dog died on the twenty-fourth day. A second adult dog which was also injected with tick emulsion developed the disease and died on the forty-third day. This was proof enough that the sporozoites contained in the tick emulsion gave the disease. This fact clearly shows that the transmission of this disease is by tick alone.

Every one knows that even if ticks are picked off a dog every day, yet a few partially engorged female ticks are found on it after some time. These ticks necessarily must be those that were thrown off from the body of another dog, either due to its scratching them off or biting them off it, before they were fully engorged. It must also be remembered that a tick remains on a dog if undisturbed for nearly five days, engorging itself the whole time with the blood of the host. The repletion of blood by a tick is at its height during the last two days or so before it leaves the host for oviposition. This gave the suggestion to see if partially engorged ticks kept starving for three or four days in a test-tube would bite a dog again to complete their engorgement with the blood of the host. The experiments proved a success.

The study of the histology of the tick shows that its pharynx opens at an angle into the buccal cavity near the ventral surface. There is a small cul-de-sac, lying behind the point of entry of the pharynx, and this is continued backwards into the œsophagus which is a short tube. The walls of the œsophagus contain radiating muscle fibres. In the proventricular fold there are thick bands of muscular tissue, and it ends abruptly at the commencement of the intestinal diverticulæ. There are slow pulsating movements in the diverticulæ. This arrangement of the mouth parts makes one believe about the

possibility to help regurgitation of blood and intestinal contents from the diverticulæ into the wound caused by the rostrum, and thence into the blood stream of the host. Again the sporozoites have movements, and it may be possible that these move into the pharynx of the tick and thence into the wound and blood stream. Some of the partially engorged infected ticks were taken and kept for four days, so as to allow enough time for the development of sporozoites. These ticks were put on two pups to which most of them readily started biting and engorging. A few of the ticks for some reason or other refused to bite. Both these pups developed the disease, one showed the parasites on the sixteenth day after being bitten by the ticks, and the other showed them on the twenty-first day, the latter showing a fairly large number of them in its peripheral blood till its death. From these experiments one feels sure that the mode of infection is through ticks. This accounts for the disease being more common in less-cared-for and tick-ridden dogs than in well-cared-for ones.

It has been observed here that inoculation of the blood from an infected dog into a healthy one does not give the disease, but inoculation of spleen puncture material successfully transmits it, the reason obviously being the presence of merozoites in it.

Symptoms of the Disease.

It is a subacute febrile disease caused by *H. canis*. The temperature ranges from 98° to 106° Farenheit, and is of an irregular oscillatory character. In the early stages the animal refuses food altogether, and later on the appetite becomes capricious. The emaciation is rapid. Pulse becomes collapsible and weak. The urine is high coloured. In the early stages there is constipation, yielding, sometimes, to diarrhœa highly tinged yellow. In advanced cases palpation of the abdomen reveals considerable enlargement of the spleen and liver, more especially of the former. The visible mucous membranes are either slightly icteric or even blanched. The disease brings about a fatal termination in from four to eight weeks. It seems to be more virulent in dogs injected with tick emulsion. During the later periods the animals become so weak that they can hardly stand. (See Fig. 1, the photograph of a naturally infected dog.)

Morbid Anatomy.

All the organs looked pale. The heart was flabby. The liver and spleen were considerably enlarged, their edges being blunt and rounded. On section the spleen pulp was seen to be softened and

dark red in colour, and the trabecular markings were distinct. The liver showed enlargement of the bloodvessels. The bone marrow was red in colour. In the dog shown in Fig. 1 the spleen weighed 2 lb. 6 oz., and the liver weighed 4½ lb., the total weight of the dog being only 26 lb.

The microscopical appearance of the section of the liver showed indistinct lobular markings. The sinuses were dilated and sparsely occupied by red blood cells. Heimosiderin pigment was seen strewn all over the section in small quantities. The cells of the liver showed fatty degeneration, though it was not limited to any zone. A few brood cysts of *H. canis* were seen. (Plate II, *C*.)

In the spleen the Malpighian bodies were indistinct in sections under the microscope, showing destruction of the lymphoid tissue. Sinuses were filled with red blood cells and plasma cells. A few endothelial cells with pigment granules were seen. The fibrous tissues of the trabeculæ showed proliferation. A large number of brood cysts were present.

In the kidneys periglomerular cell infiltration was well marked. Malpighian tufts were mostly empty. Cloudy swelling of tubules was present. The cells lining the tubules were seen to be of a low type. The lumen of the tubules contained a few blood casts. Parenchymatous degeneration of the kidneys was evident.

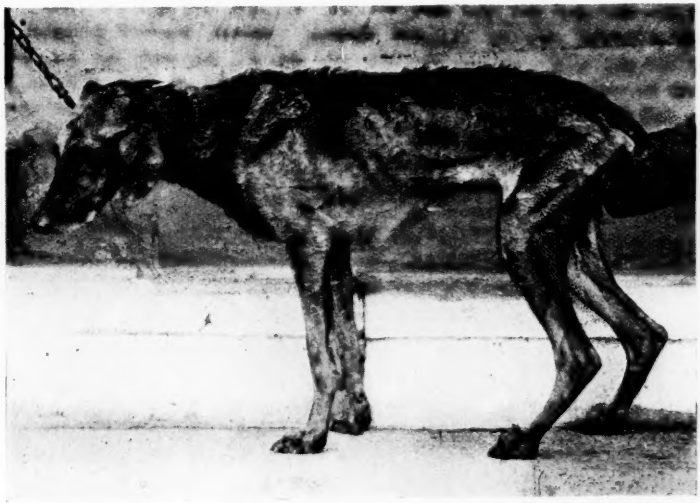

FIG. 1.—Dog Suffering from *Hæmogregarina canis*.

Diagnosis.

The disease can only be diagnosed by a careful examination of the blood under the microscope. In the early stages the changes in the blood are not marked, but in later stages the blood picture under the microscope is exactly like that obtained in a bad case of anæmia. The differential leucocyte counts in the later stages showed an increase in polymorphos and also to a degree in the basophiles. Sometimes the parasite, in the early stages, may not be encountered at all, and may lead on to a wrong diagnosis. There is no doubt, I think, that this is one of those obscure febrile conditions to which dogs are very prone in this country. The very fact that this parasite has not been reported prior to December 1921 in any of the patients here shows that it is easy to miss the parasite. In the dog from Saidapet (Case No. 216 of 22), only one parasite was seen in two smears sent, and as the disease advanced in that dog, the number of parasites also increased. In the dog whose photograph is published, over 70 per cent. of the polymorphomononuclear leucocytes were affected. In the experimental dogs, the parasites, in some stages of their development or other, have been always seen in spleen puncture fluid smears long before they appeared in the peripheral circulation. I have often taken spleen puncture fluid, for examination, from many dogs, and more than a dozen times from the same dog, without any detriment to it, and I do not see why such a course should not be adopted in all cases of fevers of obscure origin.

Treatment.

Trypan blue and salvarsan have been tried with no good results. The latter did lessen the number of sporonts in the blood for a few

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PLATE I.

A. *H. canis* in a white cell (sporont). Blood smear from an experimental dog. Highly magnified.

B. A group of *H. canis* from the blood smear from the dog shown in photograph 1. Note the number of white cells containing the parasites.

C. A "vermicule" from a hang drop. (Low magnification.)

D. Commencement of cyst formation (schizonts). The cysts are in mononuclear cells or sometimes free. Photograph of spleen puncture fluid smear.

E. A brood cyst. Smear of spleen pulp. Note the morular appearance due to the sporoblastic buds.

F. Section of a brood cyst in spleen. Note the residual protoplasm in the middle.

PLATE I.

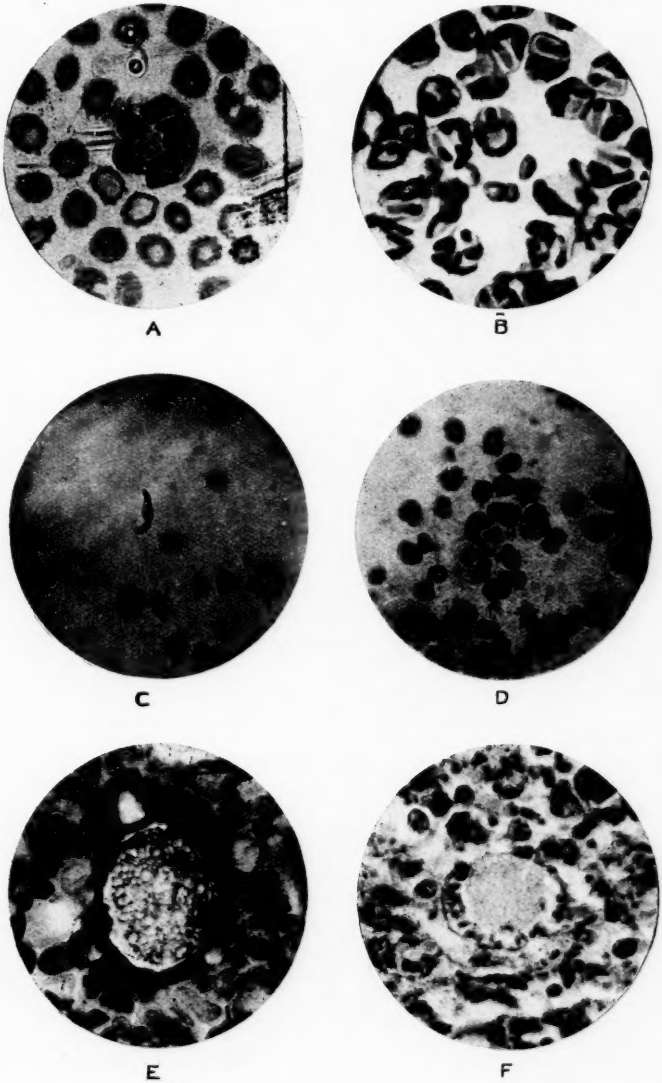
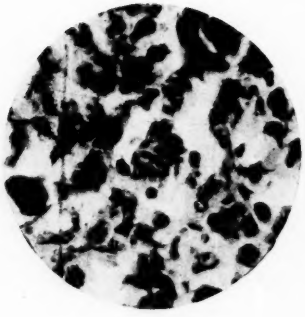


PLATE II.



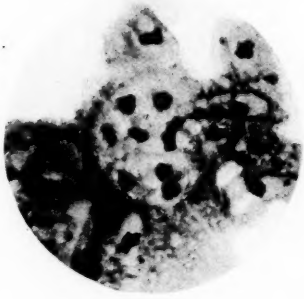
A

B

C

D

E



F

days only. Injection of tartar emetic solution has been tried only once, and the result was the same as that produced by salvarsan. Perhaps a greater number of injections may bring about a cure. Further experiments are necessary on this point, and I hope those interested will continue to work with it wherever opportunity arises. Symptomatic treatment and even careful nursing is of no avail.

Conclusion.

H. canis was first discovered several years ago, and up to now it has been looked upon as one of those parasites which cause no trouble to the host—an unlikely thing when one thinks of it. The changes in the spleen, liver, bone marrow, and kidneys in naturally infected dogs as well as in those experimentally infected are constant, and the train of symptoms are also the same. Under these circumstances, I venture to suggest that that is enough evidence to prove that there is immense harm done to the animal economy at the expense of which these organisms grow. It can be well understood by the parenchymatous changes and the periglomerular cell infiltration in the kidneys that a slow poison has been circulating in the blood, and this poison must be the toxin produced by the parasite. I may here venture to suggest that "hæmogregarinosis," if I may so call it, is a disease in dogs, and I feel confident that others will also find it to be so in course of time.

PLATE II.

A. Section of bone marrow showing merozoites. (They are in the centre of the photograph and look like a bunch of bananas.)

B. Section of the liver showing a brood cyst. Note the arrangement of the bits of nuclei as is found in the spleen. Note the degenerative changes in the hepatic cells.

C. Section of the liver showing merozoites in a brood cyst.

D. Section of the kidney showing degenerative changes in the tubules and periglomerular infiltration.

E. Secondary vermicules, in the gut of tick, about to approximate for conjugation. Note the stout and slender form.

F. Section of the gut showing sporozoites in the gut cell. Some have fallen out of the cell while cutting section. (Highly magnified.)

A CASE OF HUMAN INFESTATION WITH *BELASCARIS MYSTAX.*

By Major R. F. DICKINSON,
Royal Army Medical Corps.

The following case is of interest in that infection with this parasite is very rare in man, the optimum host being one of the Felidæ.

The patient was a girl aged three years, the daughter of British parents of good social position stationed in Mauritius. One worm was passed in a copious putty-coloured stool. The child was in good health and has remained so ever since. The fæces contained *Belascaris mystax* eggs with fine honeycomb markings.

Treatment with santonin and pulv. scammony co. on two occasions failed to produce any more worms.

A point of interest in the case is that there was a very thin and sickly cat in the house at the time from which the child probably got infected. The cat was destroyed by the child's father before the nature of its illness could be investigated.

My thanks are due to Dr. Clark H. Yeager, Senior State Director, International Health Board, Rockefeller Foundation, who very kindly cut sections of the worm and identified the specimen for me as *Belascaris mystax.—Journal of the Royal Army Medical Corps.* February 1925.

Clinical Articles

VAGINAL HÆMATOMA IN A MARE.

By W. E. ARMSTRONG, B.V.Sc., M.R.C.V.S.,
Wigton, Cumberland.

On May 21st last year I was asked to examine a mare which had given birth, under difficulties, to a dead foal five days previously. The mare was partially off her feed and the off hind leg swollen to twice its normal size as far down as the hock. The bowels were a little inactive, and the temperature 104° F., with a pulse of 72. There was no abnormal vaginal or uterine discharge. On vaginal examination a swelling was found on the right pelvic wall, and half filling the pelvic cavity. It was not painful, but soft in consistence. No definite fluctuation could be detected. I was undecided as to its nature, so I waited before opening it. An enema was given to relieve the rectum of its contents, and ʒi doses of calomel given twice daily with hyposulphite of soda and Fleming's aconite every six hours.

May 24th.—The temperature was now 102° F., and the pulse 60. The appetite was better, but the leg was more swelled, and I thought I could detect fluctuation in the pelvic swelling, so the whole surface was painted with Tinct iodi, and a sterile trocar inserted, but only a few drops of very dark blood were obtained. The calomel and the other medicine were discontinued. Exercise was advocated.

May 27th.—Fluctuation was detected, so the swelling was again painted with iodine and opened. The cavity was washed out with hot normal saline, and a pessary containing iodoform was left in the cavity. The appetite and general condition were good.

May 31st.—The leg was almost normal, the abscess cavity less and lameness almost gone. The cavity was again washed out and another iodoform pessary inserted. This was the last time I saw her. She was served a month later and is again pregnant.

TWO UNLOOKED-FOR FATALITIES.

By W. R. DAVIS, M.R.C.V.S.,
Enfield.

In March last I was asked to look at a cob that was said to be "humoury" and to have had bumps on different parts of the body and was now troubled with one on the elbow. This appeared to be only an ordinary capped elbow. I accordingly lanced it inferiorly, evacuating a quantity of serum, put in a drain of gauze soaked in solution of perchloride of mercury 1–500. I had the cob's head tied to the rack to prevent his lying down, and as he appeared to be unthrifty left a few iron and salt powders as alternatives to be given in the food.

I saw the patient a few days afterwards and all was going on well. The old drain was removed, the cavity syringed out, and a new drain inserted. Four days later on again calling I found the cob off his feed and having a high temperature and rapid pulse and exhibiting great depression. The elbow and shoulder were greatly swollen, the swelling extending inside the arm and affecting the side of the chest and abdomen.

The swelling was firm, was not emphysematous, and there was no putridity. Several incisions were made into the shoulder, and from these a small quantity only of serum, straw coloured and odourless, exuded. I ordered fomentations of hot water and lysol, gave iron quinine and chlorate of potash internally, but the cob became rapidly worse and died a couple of days afterwards. It appears that the cob had been shifted to another stall and had been allowed to lie down on a dirty floor. A smear taken from the incised tissue

showed under one-twelfth objective gram positive bacillus, which was in all probability *Bacillus Œdematicus*.

One night in February last I had a 'phone message to attend a cow at a farm seven miles away. On my arrival I found a recently calved cow with the womb completely everted. The calf was playing about the box, doubtless damaging the protruded organ. The calf having been removed, we proceeded to raise the hind part of the cow with block and tackle. The proprietor would not permit chloroform to be given as he quite expected the cow to die, or rather to require slaughtering for food. I experienced considerable difficulty in returning the organ, as the cow strained violently in spite of her raised hind parts, the pains were at times hard to control. Finally I got the mass completely returned. As I had used up all the oil I had brought with me, the farmer, who was at the cow's head, volunteered to go and get some from the house. Meanwhile I kept my arm in the womb, smoothing out the folds. I suddenly noticed that the cow was not breathing, and had her immediately lowered ; however, she was dead. The farmer declared that she was breathing all right when he let go her head. She was immediately bled, and I suppose went for beef. I am certain that the cow was not asphyxiated. The proprietor, a very experienced man, had control of the cow's head, the hind parts were not unduly raised, and bundles of straw were placed under the cow to support her. I am quite at a loss to account for the cow's death.

AN UNEXPECTED RECOVERY.

By W. R. DAVIS, M.R.C.V.S.,
Enfield.

Early in March last I was called to a cow with an inverted uterus. As the messenger informed me what the trouble was I brought pulley blocks with me. In this case also the calf was in the box with the cow, and the womb showed evidence of damage from the feet of the newborn. Having raised the cow's hind parts, chloroform was administered and the return of the organ proceeded with. Although the patient got three and a half ounces of chloroform there was considerable straining, and during one particularly severe " paining " I found that I was dealing with a portion of the peritoneal surface of the organ, and found that there was a rupture of the fundus. I immediately allowed the womb to be again everted, and got from my car a piece of very thin cotton rope, which I usually carry to make calving cords or for a twitch. I soaked the cord in ether soap and applied some of it over the uterus.

I then put a hitch of the cord round the uterus close to the vulva, and fastening a stout stick to each end of the cord got a couple of good men to tighten the hitch. When I thought this satisfactory I knotted the ends and cut off the organ an inch posterior to the ligature. The stump was well soaked with ether soap and returned. The after treatment consisted in the insertion of a salol pessary once a day for three days.

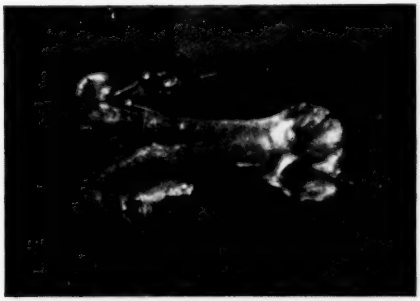
I don't think the cow was off her food for more than a day, and she is now giving four and a half gallons of milk a day and is putting on flesh.

DOUBLE VAGINA AND CERVIX IN A COW.

By W. E. ARMSTRONG, B.V.Sc., M.R.C.V.S.,
Wigton, Cumberland.

This condition is rare enough to warrant the reporting of the case, which the illustration shows none too well.

The animal, eighteen months old, was examined to ascertain the



Double Vagina and Cervix.

cause of vaginal bleeding and persistent dribbling of urine since it was six months old. The kidneys were the source of the trouble and slaughter advised.

The vagina was completely duplicated from 1 inch in front of the urethral orifice, the partition blending with the cervices anteriorly. There are two distinct cervices leading to a normal uterus and adnexa.

This case differs from those mentioned by Williams * in his excellent book, in so far as the septum was complete, and not separated from the crevices at the anterior end of the vaginæ.

* Williams, W. L., *Diseases of the Genital Organs of Domestic Animals*, 1921.

VALUE OF BONE MEAL IN CERTAIN DISEASES OF CATTLE.

By V. P. GOKHALE, G.B.V.C.,
Veterinary Inspector, Perak, Federated Malay States.

THIS short note is written with the hope that the veterinary profession will give trial to bone meal by administering it to young growing cattle in whom their development becomes stunted, to those cattle which get a plentiful supply of food but become emaciated, to those licking earth freely, and to those known to suffer (or supposed to be suffering) from parasitic gastritis, and also to other cases of emaciation from obscure causes.

The following is the history of some young animals whose growth was stunted, and of a number of emaciated cattle that were successfully treated by bone meal.

A cattle farm has been maintained at the Central Mental Hospital, F.M.S., since the latter part of the year 1913, with the object of obtaining a regular supply of good milk for the inmates, and also for obtaining manure for vegetable cultivation. They were liberally fed on grass grown on the farm and were given a ration of conjee made up of broken rice and rice bran, with an addition of small quantity of salt and oil cake. They were kept in well-ventilated clean stalls having concrete flooring and looked after well. They appeared to be growing splendidly till about the June of 1922. At this period there were sixty-seven head of cattle (calves not included), and a few of them seemed running down in condition. Tonic powders made up of ferri sulph, nux vomica, and gentian, were administered without benefit. Gradually the number of emaciated cattle increased, the young did not seem to grow normally, and non-contagious abortions, retention of placentas, and unusually high mortality among calves, became of frequent occurrence. Later, they freely indulged in licking earth, had poor appetite, appeared dull, and used to grind their teeth. Examination of the blood for parasites proved negative. Liquor arsenicalis was given a trial, and as they showed improvement in appetite and condition it was administered for over two months; but the old trouble started again among the treated animals, and so it was discontinued. At this stage it was thought advisable to destroy some more advanced cases to ascertain the cause on post-mortem examination. Five such cases were destroyed, and on post-mortem examination the only important finding was parasitic gastritis of advanced type and a large quantity of sand present in the abomasum. Solution of copper sulphate was administered to the remaining emaciated cattle. This treatment proved much more promising than the arsenic treatment, but the improvement was of short duration

only, as the treated cattle again began to lose condition rapidly. The whole situation became now of an alarming nature as, from June 1922 up to the end of October 1924, no less than twenty-three young adult cattle had died and five hopeless animals were destroyed for investigation. At this critical juncture the G.V.S., Perak North, Captain G. Moir, O.B.E., F.R.C.V.S., happened to receive a pamphlet entitled " Phosphorus in the Live Stock Industry," reprinted from the *Journal of the Department of Agriculture,* May 1924; and after its perusal thought to give a trial to bone meal; and so 3 ounces of bone meal, mixed with a pint of conjee, was administered as a drench to each animal daily. Twenty-seven head of cattle were thus treated. Under this treatment they began to show improvement rapidly, and within less than three months they put on flesh, the grinding of teeth and habit of licking earth disappeared, and their appetite became normal. They now appear to be in the best of heath and condition.

Concluding remarks :—

(1) It ought to be mentioned here that no death has occurred from the commencement of the experiment by bone meal till the present date.

(2) It will be seen from the above that as a matter of fact the trouble was not due to infestation of *Hæmonchus contortus* as the copper sulphate treatment was a total failure, whereas bone meal alone brought on the improvement. It is obvious that chronic parasitic gastritis became prominent owing to impaired health of the cattle.

(3) As bone meal alone proved successful where treatment with drugs failed, there is a strong reason to believe that the grass grown on the farm during recent years was highly deficient in phosphorous content, obviously from deterioration of the soil by age and lack of treatment by suitable manures. Unfortunately analysis of the soil and fodder was not undertaken owing to lack of facilities, and therefore a definite statement of any sort cannot be made.

However, judging from the results obtained, there does not seem anything to doubt the above conclusions.

(4) From the results obtained it can be easily understood that in suitably nourished animals the harm that may be caused by parasites in debilitated and ill-nourished animals is reduced to a minimum.

(5) It must be again emphasised that proper animal nutrition is indisputably one of the chief factors in the prevention of disease.

(6) Bone meal supersedes other phosphorus preparations owing to its power of easy assimilation, cheap price, being easily obtainable, and non-toxic.

(7) Bone meal has been experimentally found as most economical for fattening cattle for beef, as by its regular administration the assimilation of food is greatly increased and thus obtains a better value for the food given.

(8) It is most important to remember that bone meal should be obtained from a reliable source and must have been sterilised, as the use of Indian bone meal as a farm fertiliser recently caused the death, in Scotland, of two horses from anthrax.

I hope the admirable pamphlet on "Phosphorus in the Live Stock Industry" will be read carefully by those who have not seen it yet, as it contains many important and interesting facts about assimilation and metabolism in cattle when they are given bone meal.

Abstracts of Current Literature

Saint-Cyr, M.—Urethral Obstruction of Verminous Origin in the Dog.—*Société des Sciences Vétérinaires*, June 1, 1924.

A SPORTING dog out in a well-watered district went to a pond to drink and afterwards presented a complete urethral obstruction. Urethrotomy revealed the presence in the canal of a *Eustrongylus renalis gigas*, measuring 20 inches long and 1¼ inch wide. Cure after extraction was rapid and complete in a week.

The animal never having shown any pain or symptom previously, the author thinks that the parasite may have developed in the bladder, and not as usually in the kidney.

Escoffier, M.—The Treatment of Diarrhœa by Basic Salicylate of Alum.—*Société des Sciences Vétérinaires*, March 30, 1924.

SALICYLATE of alum is a new drug finding its place as a useful astringent. In an alkaline medium it is slowly decomposed, forming a veritable absorbent jelly of alum, whilst the salicylic acid liberated acts antiseptically and slowly dialyses. The drug contains:—

Acid salicylic	..	..	68·8 per cent.
Alum anhydrous	..	..	26·5 ,, ,,

It is not toxic. It possesses a double property, being astringent and antiseptic. It is effective in motor, secretory, or sensory trouble of external or internal origin. It is useful in diarrhœa and enteritis in the horse, cow, and dog. Its effect is constant and quick. In the dog severe diarrhœa is checked in four to six days. There are

no contra-indications, and it can be used quite well in combination with other drugs, notably opium and belladonna. The dose for the horse is 2 to 6 drachms; for the dog 15 to 45 grains. These doses can be varied safely within large limits and without danger. It is a whitish or rose coloured powder, insoluble in water and tasteless, and much cheaper than sub-nitrate of bismuth.

Mayer, V.—Coccidiosis in Dogs.—*Vet. Med. Inang. Diss., Vienna*, 1922; *B.T.W.*, No. 18, May 1st.

ACCORDING to the author, four cases were treated which were marked by obstinate diarrhœa. The fæces were brownish green to greenish black, watery, cadaverous smelling, and containing much mucus. In 248 tests of the fæces of dogs, which were affected with various internal and skin diseases, the eggs of the following parasites were found: round worms, 68 per cent.; flat worms, 12·1 per cent.; coccidia, 1·6 per cent.; and free from parasites, 17·7 per cent.

Schiel of Jever.—Cure of Catarrh of the Guttural Pouches by Extirpation.—*B.T.W.*, No. 18, May 1st.

THE swelling in the region of the ear glands due to a bulging of the air sac by gas, fluid, and hard masses as a result of catarrhal disease of its mucous membrane, is easily discernible clinically. Air sac and mucous membrane then form a hard membrane. These peculiar catarrhal accretions of the air sac are very difficult to cure, since despite operative removal of the contents and treatment of the mucous membrane with astringents after closure of the wound recurrences generally occur.

In a yearling foal after opening up several times and long continued treatment the swelling always appeared again. Since the animal was falling off in condition the owner requested me to operate. A left-sided catarrh of the air sac was present. After a long cutaneous incision behind the parotid gland at the point of the swelling, I dissected out the pouch and then separated the hard membrane of the sac with my fingers from its surroundings, and by this means made three-quarters of the membrane accessible. This separated portion of the membrane was then cut off with the scissors; the remainder was left *in situ*. Recovery went steadily on, and after four weeks the foal recovered. I have had the animal under observation up to four years old. The horse seems in no way depreciated, there has been no disturbance of respiration and no difficulty in swallowing. I am recording this case because of its rarity in practice, and hope others meeting with a like condition will test it, since individual practitioners can scarcely have a great experience of it.

Sambon, L. W.—Researches on the Epidemiology of Cancer made in Iceland and Italy (July–October, 1924).—*Journ. Trop. Med. and Hyg.*, xxviii, No. 3, pp. 39–71, 33 figs. London, February 2, 1925.

THE author briefly reviews data collected in Iceland and Italy concerning the local occurrences of cancer [cf. *R.A.E.*, B, xii, 128]. These facts all point to the importance of parasitic worms in relation to the disease, in which connection *Muspicea borreli*, gen. et sp. n., is described from Strasbourg taken in the subcutaneous connective tissue of the inguinal glands of a mouse. The restriction of cancer to definite streets, even houses, is again demonstrated. In all such cases the intermediate hosts (cockroaches, etc.) of the parasitic worms were present. The predominant species in Iceland is *Blattella germanica*, L. It is believed that these observations in certain districts of Iceland and Italy prove the usefulness of intensive scientific research in limited areas of different countries where the disease is most prevalent and indicates the uselessness of statistics, such as are compiled in central official bureaux by mixing and blending data of very different importance and value, gathered from innumerable localities, and losing precious facts in the resulting confusion. Whatever the rôle of parasitic worms, the actual essential cause of cancer is some ubiquitous micro-organism, probably a filter-passer, as the researches of Rous and his fellow workers seem to indicate, but this parasite, apparently, is unable to thrive except in tissues of lowered resistance.—*Review of Applied Entomology.*

Leiper, R. T., F.R.S, and Cameron, T. W. M., M.A., B.Sc., Ph.D., M.R.C.V.S.—Demonstration of Some of the Pathological Results of Helminthic Infections in Animals.—*Proceedings of Royal Society of Medicine*, vol. xvii, No. 9, July 1924.

THE authors pointed out that lesions due to worm parasites were liable to occur in any part of the body. Thus in the respiratory tract there is found *Oslerius osleri*—a small filarid worm—which causes nodule formation on the trachea of the dog; and various species of Paragonimus in the lungs of carnivores and omnivores which result in the formation of verminous cysts. In the intestinal tract many helminths occur. Several examples were exhibited: (*a*) *Spirocerca sanguinolenta*, forming large tumours in the stomach of the dog; (*b*) *Œsophagostomum radiatum*, the cause of " pimply gut " or nodular disease of the intestine in cattle; (*c*) *Echinorhynchus gigas*, perforating the intestine of pigs and causing peritonitis; (*d*) *Anoplocephala perfoliata*, responsible for serious ulceration of the large intestine of horses. Outside of the alimentary and respiratory tracts there were

also found lesions caused by helminths. The examples shown were: (*a*) *Dioctophyme renale*, destroying the kidney substance in dogs; (*b*) *Dirofilaria immitis*, occluding the heart in the same host; (*c*) *Eleophora poeli*, in nodules, and causing aneurysms in the aorta of bovines; (*d*) *Onchocerca*, causing worm nests in Australian cattle. Pathological specimens illustrating the lesions caused by these parasites were shown. Professor Leiper pointed out that these were only a very few of the conditions caused by helminths, and that the importance of the parasites in animals and human pathology was generally very much under-estimated.

Notices

AGRICULTURAL AND VETERINARY RESEARCH SCHOLARSHIPS.

THE Ministry of Agriculture and Fisheries invites applications for research scholarships in agricultural and veterinary science. The number to be awarded will depend upon the qualifications of the candidates and will not in any case exceed seven. The scholarships are tenable for three years from October 1, 1925, and are of the value of £200 per annum. Applications must be received not later than June 30, 1925, on the prescribed form (900/T.G.) which, together with a copy of the conditions attaching to the scholarships, may be obtained from the Secretary, Ministry of Agriculture and Fisheries, 10 Whitehall Place, London, S.W. 1.

INTERNATIONAL HORSE SHOW AT OLYMPIA.

THE Fourteenth International Horse Show will be held at Olympia from June 22nd to 30th under the patronage of His Majesty the King. There are about two thousand entries, and the judging will take place all day long, the public being allowed to be present on each occasion. The usual varieties of jumping tests have been arranged for, and representatives from Belgium, France, Great Britain, Holland, Italy, Poland, and Sweden have already sent in their entries for the King George V Gold Cup which is at present held by Italy. The competition for this will take place on the opening day, and the test for the Prince of Wales's Cup will be made on the afternoon of the 26th. Last year this trophy was won by British cavalrymen.

Seats can be booked in advance, either at Olympia or at the International Horse Show office, 12 Hanover Square, London, W. 1.

MODEL ABATTOIR AT WEMBLEY EXHIBITION.

In the Ministry of Health Section at the British Empire Exhibition at Wembley there is an up-to-date illustration of a model abattoir, and it is placed there in the hope that it will be examined by Local Authorities who have the wish to undertake the construction of public abattoirs. The offices of the Model Abattoir Society are at the Royal Sanitary Institute, 90 Buckingham Palace Road, London, S.W. 1, and inquiries will be welcomed from all members of the veterinary profession.

The King's Birthday Honours

Readers of The Veterinary Journal and old students of the London Veterinary College will be pleased to see the name of Sir John Bland Sutton in the King's Birthday Honours List as the recipient of a baronetcy.

Sir John's support of our profession, and of Comparative Medicine in particular, is well known, and at one time he held the Lectureship of Pathology in the Royal Veterinary College, London.

Obituary

MR. PERCY GREGORY, M.R.C.V.S., TONBRIDGE.

One of the best and most popular of the South of England veterinary practitioners, in the person of Mr. Percy Gregory, M.R.C.V.S., of Tonbridge, passed away on May 5th, as he would have wished, literally whilst in harness. Born of a veterinary family, and educated at Tonbridge School, Mr. Gregory entered the profession early, graduating from the London College in 1877. Acting for a time as assistant in conjunction with his father, he eventually succeeded to the practice, and his word was respected not only amongst a large personal *clientèle*, but also by his brother practitioners in the County of Kent. Always a strong supporter of anything which was likely to lead to the welfare of the profession, he served as a Vice-President of the South-Eastern Branch of the National Veterinary Association, whilst his public official duties included those of Veterinary Inspector to the Kent County Council and a number of posts connected with Tonbridge itself. His genial presence will be much missed by a large circle of friends, and the respect in which he was held was fully demonstrated by the large following of clients and others who attended his funeral.